

CASE REPORT

Impella-assisted transcatheter closure of an acute postinfarction ventricular septal defect

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SUMMARY

This case report describes a 72-year-old woman who developed an acute postmyocardial infarction ventricular septal defect (VSD) with consequent cardiogenic shock. Intra-aortic balloon pump (IABP) counter-pulsation was urgently initiated in the cardiac catheterisation laboratory, with neither clinical nor haemodynamic improvement, prompting immediate removal of the IABP and the insertion of an Impella 2.5 heart pump (AbioMed Inc; Danvers, Massachusetts, USA), a temporary ventricular assist device. Thereafter, the patient improved clinically and was admitted to the cardiovascular intensive care unit (ICU). While in the cardiovascular ICU, the patient developed worsening mechanical haemolysis of blood cells, stable but persistent cardiogenic shock and a transient ischaemic attack. A consensus decision was made to proceed with percutaneous repair of the VSD as she was deemed at high risk for surgical repair. She underwent successful percutaneous VSD repair on day 4 of hospitalisation, using a single 18 mm Amplatzer muscular VSD occluder (AGA Medical, Plymouth, Minnesota, USA) with trace residual flow across the occluder. Adequate systolic blood pressure and cardiac output was maintained postprocedure with the Impella 2.5 device. The patient, however, succumbed to multiorgan dysfunction occasioned by sepsis.

BACKGROUND

Postinfarction ventricular septal defect (VSD) is a rare but serious event occurring in 0.2% of all infarcts and 2–5% of patients presenting with cardiogenic shock.¹ It is associated with 50% mortality after surgical repair.^{2–6} In the pre-fibrinolytic era, VSD was commonly seen 3–7 days after myocardial infarction (MI), but is now generally diagnosed within the first 24 h after an MI.⁷ Although the use of an Impella heart pump device as a bridge to surgical VSD repair and heart transplantation has been reported in the literature,^{8–10} Impella-assisted percutaneous repair of postinfarction VSD in high surgical risk patients has not been described. We report a rare case of immediate postinfarct VSD in a very high surgical risk patient who underwent successful percutaneous closure using an Impella 2.5 heart pump device.

CASE PRESENTATION

A 72-year-old woman with a history of hypertension and coronary artery disease status postcoronary bypass surgery 10 years prior and status postpercutaneous coronary intervention (PCI) with stent to the mid left anterior descending coronary

artery for an atretic internal mammary artery graft 2 years later, was transferred from an outside hospital, with acute ST-elevation MI, after she presented with substernal chest tightness radiating to her left shoulder. At the outside hospital, her initial troponin had been negative and ECG unremarkable. Owing to continued chest pain, a repeat ECG was performed, which revealed ST-elevation in leads I, aVL and V2, and a rise in troponin to 0.7 ng/mL, prompting urgent transfer for primary revascularisation. Physical examination revealed a grade 2/6 holosystolic murmur at the left lower sternal border with radiation across the precordium and clinical features of congestive heart failure and cardiogenic shock. Her systolic blood pressure was 62 mm Hg. She was started on an intravenous dopamine drip with subsequent insertion of an intra-aortic balloon pump (IABP). She underwent an urgent and successful PCI with stenting of the mid left anterior descending. Left ventricular angiography revealed an ejection fraction of 25% with no other significant finding. Postintervention, the patient remained hypotensive with systolic blood pressure <90 mm Hg. To improve her haemodynamics while she was still in the cardiac catheterisation laboratory (CCL), the IABP was exchanged for an Impella 2.5 device, with improvement in haemodynamics and systolic blood pressures above 90 mm Hg off pressors. A two-dimensional (2D) echocardiogram in the CCL ([figure 1](#)) revealed a 1.5 cm apical postinfarction VSD, a left ventricular ejection fraction of 25–30% and mildly reduced right ventricle systolic function. There was left-to-right shunting across the VSD, with a gradient of 36 mm Hg and QP/QS ratio >1.8. Although uncommon in the pre-fibrinolytic era, post-MI VSD is now generally diagnosed within 24 h.⁷ Our patient underwent urgent PCI at the onset because VSD was not diagnosed until later, when 2D echocardiogram was performed for persistent haemodynamic compromise. She was referred for surgical VSD repair but ultimately turned-down for surgery due to very high operative risk. While in the cardiovascular intensive care unit (ICU) on Impella 2.5 support, she developed worsening hepatic congestion and renal failure, pancytopenia from mechanical haemolysis and transient ischaemia attack despite optimal systolic blood pressure. A decision was made to proceed with urgent percutaneous closure of the VSD to potentially reverse the worsening clinical course. Under general anaesthesia, an intraoperative transoesophageal echocardiogram was performed, confirming the findings noted above. An 18 mm Amplatzer muscular VSD



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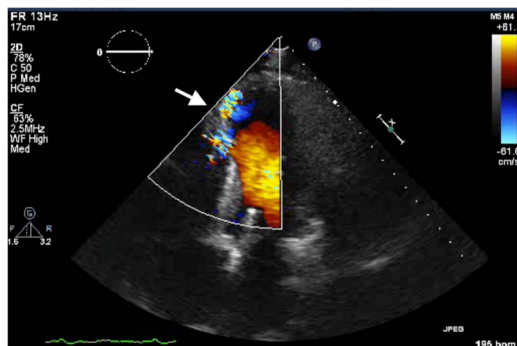


Figure 1 Transthoracic echocardiography four-chamber view of the postmyocardial infarction ventricular septal rupture (arrow).

occluder (AGA Medical, Plymouth, Minnesota, USA) was deployed across the VSD in standard technique under fluoroscopic and echocardiographic guidance through a 9 Fr TorqVue 180 delivery system (AGA Medical) placed via the right internal jugular vein. Postdeployment echocardiogram revealed trace residual shunt across the repaired VSD (figure 2) and fluoroscopy revealed an optimally positioned device (figure 3).

OUTCOME AND FOLLOW-UP

The Impella 2.5 device was left in situ for haemodynamic support and the patient transferred to the ICU. She made an initial clinical improvement, but ultimately succumbed to multi-organ dysfunction triggered by sepsis.

DISCUSSION

Transcatheter closure of a postinfarct VSD is a high-risk procedure with a high failure rate.¹¹ In patients who are haemodynamically unstable and deemed high risk for surgery, transcatheter closure remains the only alternative to medical management, which has an almost 100% mortality rate.¹ Although, transcatheter closure of acute postinfarction VSD is increasingly being performed,^{12 13} there are several associated potential risks due partly to the poor clinical condition of the patients and the ill-defined and friable margins of the rupture, which may lead to incomplete closure or device embolisation. Poor prognostic factors following VSD include presence of cardiogenic shock, right ventricular dysfunction and inferior infarct.^{14–17} Although Szkutnik *et al*¹⁸ suggested in their series that percutaneous VSD closure should be performed after the sixth postinfarction week when the scar tissue is more solid and therefore better defined, delayed VSD closure in haemodynamically unstable patients

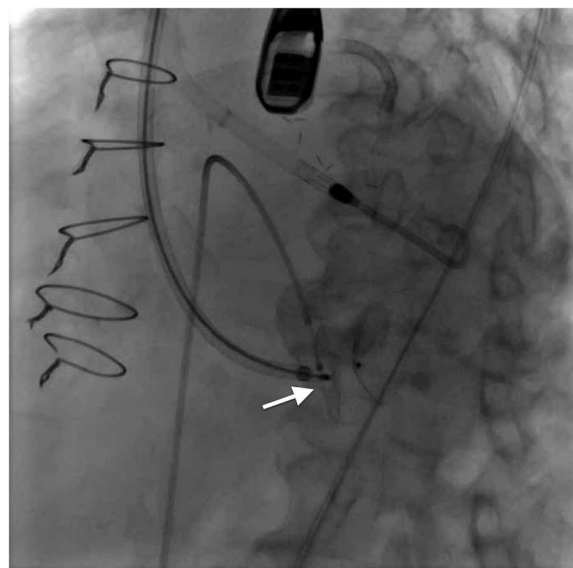


Figure 3 Fluoroscopy showing an optimally positioned Amplatzer device (arrow).

usually results in progressive multisystem failure with very poor prognosis. The onset of complications such as mechanical haemolysis, pancytopenia and transient ischaemic attack while on the Impella heart pump device, as previously described in the literature,¹⁹ can pre-empt immediate rather than delayed closure of the VSD, as was the case in our patient. Also, the current guidelines recommend immediate surgical VSD closure irrespective of the patient's haemodynamic condition.^{20 21} Our patient was considered a non-surgical candidate at the onset due to cardiogenic shock and severe left ventricular dysfunction. The decision to proceed with percutaneous closure was taken in the absence of any other alternative except medical management with its attendant bleak outcome. The patient underwent successful transcatheter closure of her VSD with Impella heart pump support, ultimately succumbing to progressive multiorgan dysfunction.

Conclusion

In patients with postinfarction VSDs who are not candidates for surgical closure or those who have haemodynamically significant residual shunts, Impella-assisted transcatheter closure offers an important therapeutic option versus an otherwise grim outcome

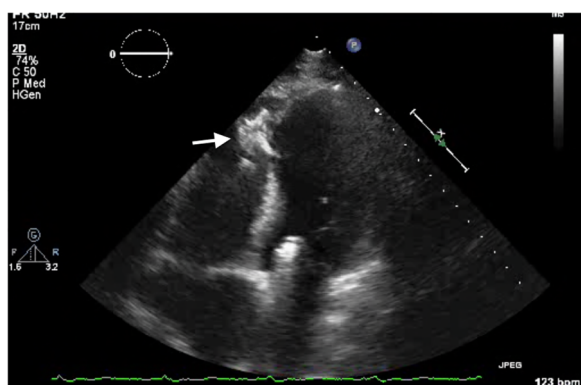


Figure 2 Transthoracic echocardiogram showing successfully repaired ventricular septal rupture with an Amplatzer device (arrow).

Learning points

- ▶ Postinfarction ventricular septal defect occurs in 2–5% of patients with cardiogenic shock.
- ▶ Mortality approaches 50% with surgical repair and 100% in patients treated medically.
- ▶ Percutaneous closure of the defect is a viable alternative in very high surgical risk patients.
- ▶ The Impella 2.5 device ensures optimal haemodynamics during percutaneous closure of a defect.
- ▶ In spite of optimal results with transcatheter closure, patients with a postinfarction ventricular septal defect represent a group of critically ill patient who may experience adverse outcomes from other associated comorbidities.

without treatment. Although current guidelines recommend immediate surgical closure of a postinfarction VSD,²⁰ timing of surgery remains controversial and sometimes contra-indicated in the acutely unstable patient with prohibitive risk. Transcatheter closure, although effective in elective closure of VSD, is a high-risk procedure with high likelihood of failure in the acute post-infarction period.¹¹

Competing interests None declared.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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